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     (FILE 'HOME' ENTERED AT 10:37:11 ON 17 MAR 2004)

     FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, CANCERLIT, JAPIO' ENTERED AT
     10:37:29 ON 17 MAR 2004
L1          21773 S NEURITE? AND OUTGROWTH?
L2             40 S L1 AND LUMINES?
L3            391 S NEUROFILAMENT AND IMAGE?
L4              1 S L2 AND L3
L5             25 S L3 AND L1
L6             12 DUPLICATE REMOVE L5 (13 DUPLICATES REMOVED)
L7             11 S L6 NOT L4
L8           2359 S L1 AND NUCLE?
L9              2 S L8 AND L3
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ANSWER 3 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1999:198128 CAPLUS
DN 131:16923
ED Entered STN: 29 Mar 1999
TI Effects of different fragments of tenascin-R on neuron morphology in vitro
AU Xu, Hanpeng; Xiao, Huasheng; Wang, Haojun; Liang, Zhe; Gong, Ju
CS The Institute of Neuroscience, Fourth Military Medical University, Xi'an, 710033, Peop. Rep. China
SO Journal of Medical Colleges of PLA (1998), 13(4), 272-275
CODEN: JMCPE6; ISSN: 1000-1948
PB Journal of Medical Colleges of PLA, Editorial Board
DT Journal
LA English
CC 13-6 (Mammalian Biochemistry)
AB To investigate the effects of different tenascin-R fragments on morphol. changes of neurons in vitro, cell suspension were prepared from E14-15 mouse embryo spinal cords by mech. dissection and trypsin digestion. The cells were cultured in dishes coated with different bacterial expressed tenascin-R fragments. After being cultured in serum-free medium for 26 h, the cells were fixed and stained by ABC immunocytochem. method for NSE. The cell number and **neurite** length were measured by a stereol. method using an **image** anal. system, and the data was analyzed statistically. The cells grew well in the serum-free medium for 26 h. Three types of cells were identified: (1) phase-bright cells with single or double **neurites**; (2) phase-dark cells with well branching **neurites**; (3) flat cells with 2-4 round vesicles in the **cell body** and radio-like **neurites**. The cell number and the **neurites** length were influenced by different tenascin-R fragments. It was found that FN1-2 fragment inhibited **neurite outgrowth**. Different tenascin-R fragments that were used as substrate exert varying effects on cultured neural cells, adhesion or anti-adhesion of cells, promotion or inhibition of the growth of **neurites**. These influences were mediated through receptors on the cell membrane. This study may provide some clues to the search for the search for the different receptors which may play essential roles in neuronal development and plasticity.
ST tenascin R neuron morphol
IT Nerve
(neuron; tenascin-R fragment effect on neuron)
IT Axon
(**outgrowth**; tenascin-R fragment effect on neuron)
IT Cell adhesion
Cell morphology
Spinal cord
(tenascin-R fragment effect on neuron)
IT Tenascins
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(tenascin-R; tenascin-R fragment effect on neuron)
RE.CNT 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD
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ANSWER 5 OF 7 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1998:517015 CAPLUS
DN 129:273372
ED Entered STN: 20 Aug 1998
TI p21RhoA and p21RhoA binding proteins as regulators of lysophosphatidic acid (LPA)-induced changes in neuronal morphology
AU Gibbink, Martijn F. B. G.; Kranenburg, Onno; Jalink, Kees; Postma, Friso R.; Poland, Mieke; Houssa, Brahim; Oomen, Lauren; Van Horck, Francis P. G.; Moolenaar, Wouter H.
CS Divison Cellular Biochemistry, The Netherlands Cancer Institute, Amsterdam, 1066 CX, Neth.
SO Kinases and Phosphatases in Lymphocyte and Neuronal Signaling (1997), 235-241. Editor(s): Yakura, Hidetaka. Publisher: Springer, Tokyo, Japan. CODEN: 66NHAY
DT Conference; General Review
LA English
CC 13-0 (Mammalian Biochemistry)
AB This is a **review** with 34 refs. Addition of lysophosphatidic acid (LPA), sphingosine-1-phosphate or thrombin to serum-deprived N1E-15 neuronal cells results in rapid neurite retraction and rounding of the **cell body**. These morphol. changes are accompanied by rapid assembly of filamentous actin and contraction of the cortical actin cytoskeleton. Treatment of the cells with Clostridium botulinum C3 exoenzyme, which ADP-ribosylates and thereby inactivates the Rho small GTP binding proteins, inhibits agonist induced cell rounding. Furthermore, dominant neg. RhoA stimulates cell flattening and **neurite outgrowth** similar to C3 toxin. Cells expressing dominant-neg. RhoA also fail to change shape in response to LPA. Activated V14RhoA mimics LPA action in inducing cell rounding and inhibiting neuronal outgrowth. To further elucidate the signaling mechanisms that regulate RhoA mediated changes in neuronal morphol., novel RhoA binding proteins were identified. RhoGEF is a novel GDP/GTP exchanger that interacts with both wild-type and activated V14RhoA, but not with Rac or CDC42. Similar to activated V14RhoA, RhoGEF induces cell rounding. P116Rip is a novel RhoA binding protein whose overexpression stimulates **neurite outgrowth**. These results establish RhoA, RhoGEF and p116Rip as critical determinants of RhoA-mediated neuronal shape changes. Identification of proteins that regulate LPA induced actin cytoskeletal remodeling will help to understand to intracellular mechanisms by which neuronal cells regulate their complex morphol.
ST **review** p21RhoA protein neuronal morphol regulator; p116Rip RhoGEF protein neuronal morphol **review**
IT Proteins, specific or class
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(RhoGEF; p21RhoA and p21RhoA binding proteins as regulators of lysophosphatidic acid (LPA)-induced changes in neuronal morphol.)
IT Nerve
(neuron; p21RhoA and p21RhoA binding proteins as regulators of lysophosphatidic acid (LPA)-induced changes in neuronal morphol.)
IT Proteins, specific or class
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(p116Rip; p21RhoA and p21RhoA binding proteins as regulators of lysophosphatidic acid (LPA)-induced changes in neuronal morphol.)
IT Cell morphology
Cytoskeleton
(p21RhoA and p21RhoA binding proteins as regulators of lysophosphatidic acid (LPA)-induced changes in neuronal morphol.)
IT Lysophosphatidic acids
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(p21RhoA and p21RhoA binding proteins as regulators of lysophosphatidic

acid (LPA)-induced changes in neuronal morphol.)
IT Rho protein (G protein)
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(p21rhoA; p21RhoA and p21RhoA binding proteins as regulators of lysophosphatidic acid (LPA)-induced changes in neuronal morphol.)
RE.CNT 34 THERE ARE 34 CITED REFERENCES AVAILABLE FOR THIS RECORD
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     (FILE 'HOME' ENTERED AT 11:17:43 ON 17 MAR 2004)

     FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, CANCERLIT, JAPIO' ENTERED AT
     11:18:09 ON 17 MAR 2004
L1           3714 S (CELL BOD?) AND NEURITE?
L2            538 S L1 AND NUCLE?
L3            113 S L2 AND OUTGROWTH?
L4              4 S L3 AND IMAGE?
L5              1 DUPLICATE REMOVE L4 (3 DUPLICATES REMOVED)
L6            710 S (NEURITE OUTGROWTH) AND REVIEW
L7              0 S L6 AND IMAGE?
L8              8 S L6 AND (CELL BOD?)
L9              7 DUPLICATE REMOVE L8 (1 DUPLICATE REMOVED)
L10         38031 S FLUORES? AND LUMINES?
L11          1055 S L10 AND REVIEW?
L12            30 S L11 AND IMAGE?
L13            25 DUPLICATE REMOVE L12 (5 DUPLICATES REMOVED)
L14             0 S (FLUORE? VERSES LUMINES?)

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L7            0 S L6 AND IMAGE?
L8            8 S L6 AND (CELL BOD?)
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L12          30 S L11 AND IMAGE?
L13          25 DUPLICATE REMOVE L12 (5 DUPLICATES REMOVED)
L14           0 S (FLUORE? VERSES LUMINES?)

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Developmental Brain Research
Volume 108, Issues 1-2 , 15 June 1998, Pages 205-216

doi:10.1016/S0165-3806(98)00050-9 Cite or link using doi

Research report

Depolarization stimulates lamellipodia formation and axonal but not dendritic branching in cultured rat cerebral cortex neurons

G. J. A. Ramakers*, J. Winter, T. M. Hoogland, M. B. Lequin, P. van Hulten, J. van Pelt and C. W. Pool

Netherlands Institute for Brain Research, Graduate School Neurosciences Amsterdam, Meibergdreef 33, 1105 AZ Amsterdam ZO, Netherlands

Accepted 3 March 1998. Available online 23 December 1998.

Abstract

Electric activity is known to have profound effects on growth cone morphology and neurite outgrowth, but the nature of the response varies strongly between neurons derived from different species or brain areas. To establish the role of electric activity in neurite outgrowth and neuronal morphogenesis of rat cerebral cortex neurons, cultured neurons were depolarized for up to 72 h and quantitatively analyzed for changes in axonal and dendritic morphology. Depolarization with 25 mM potassium chloride induced a rapid increase in lamellipodia in almost all growth cones and along both axons and dendrites. Lamellipodia formation was dependent on an influx of extracellular calcium through L-type voltage-sensitive calcium channels. Prolonged depolarization for 24 h induced an increase in total axonal length, mainly due to an increase in branching. After three days of depolarization axonal outgrowth was largely the same as in control neurons, suggesting accommodation of the growth cones to chronic depolarization. Dendrites showed very little change during the first three days in culture, and dendritic length or branching were not affected by depolarization. Thus, in early cerebral cortex neurons depolarization specifically stimulates axonal outgrowth through increased branching. This increase in branching may be a consequence of the earlier increase in lamellipodia formation. In contrast, early dendrites seem to be unable to translate the increase in lamellipodia into changes in outgrowth or

branching. This difference between axons and dendrites could be due to differences in the stabilization of the tubulin cytoskeleton.

Author Keywords: Neuronal morphogenesis; Growth cones; Depolarization; Neurite outgrowth and branching; Electric activity

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Developmental Brain Research
Volume 108, Issues 1-2 , 15 June 1998, Pages 205-216

ANSWER 20 OF 34 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1998:699079 CAPLUS
DN 130:61163
ED Entered STN: 04 Nov 1998
TI Nerve growth factor-responsive, transcription-independent outgrowth of neurites in a clonal variant of PC12 cells (PC12D)
AU Sano, Mamoru
CS Aichi Human Service Center, Institute for Developmental Research, Aichi, 480-03, Japan
SO Current Topics in Neurochemistry (1997), 1, 27-40
CODEN: CTNEFZ
PB Research Trends
DT Journal; General Review
LA English
CC 2-0 (Mammalian Hormones)
AB A **review**, with 119 refs. Most studies of the NGF-dependent outgrowth of neurites have been performed with cultured sympathetic and sensory neurons or PC12 cells. The primary neurons have had prior exposure to NGF in vivo and they require NGF for basic survival. Although PC12 cells do not require NGF for survival, the outgrowth of neurites in such cells is a consequence of their differentiation into cells that resemble sympathetic neurons in response to NGF. Thus, the outgrowth of neurites is not necessarily a direct consequence of exposure to NGF. PC12D cells, a stable variant subcloned from native PC12 cell populations, produce neurites in a rapid transcription- and translation-independent manner upon exposure to NGF, bFGF, dbcAMP or staurosporine. The rapid sprouting of neurites occurs within minutes in local regions of PC12D cells that are exposed to NGF. Recent studies in conventional PC12 cells demonstrated the close relationship between the activation of MAP kinases (ERKs) and the outgrowth of neurites in response to various agents. Simultaneous activation and rapid **nuclear** translocation of MAP kinases were also observed in PC12D cells treated with NGF or bFGF. But the activation of MAP kinases was not observed in the outgrowth of neurites induced by dbcAMP or staurosporine. In this cell line, the NGF-dependent outgrowth of neurites was not blocked by the inhibition of the activation of MAP kinases by a MEK inhibitor, PD-98059. These results indicate that the activation of MAP kinases and subsequent expression of specific genes are required for the NGF-induced differentiation of PC12 cells but this pathway is not required for the NGF-dependent outgrowth of neurites. These processes have been investigated together in most studies of conventional PC12 cells. PC12D cells provide a unique exptl. system for studies of the cellular mechanism of the NGF-induced sprouting and elongation of neurites, sep. from the transcription-dependent differentiation of the cells.
ST **review** NGF transcription **neurite outgrowth**
PC12 neuron signaling differentiation
IT Animal cell line
(PC12; nerve growth factor-responsive and transcription-independent outgrowth of neurites in clonal variant of PC12 cells)
IT Nerve
(differentiation; nerve growth factor-responsive and transcription-independent outgrowth of neurites in clonal variant of PC12 cells)
IT Signal transduction, biological
Transcription, genetic
(nerve growth factor-responsive and transcription-independent outgrowth of neurites in clonal variant of PC12 cells)
IT Cell differentiation
(neuronal; nerve growth factor-responsive and transcription-independent outgrowth of neurites in clonal variant of PC12 cells)
IT Axon
(outgrowth; nerve growth factor-responsive and transcription-

ANSWER 20 OF 34 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1998:699079 CAPLUS
DN 130:61163
ED Entered STN: 04 Nov 1998
TI Nerve growth factor-responsive, transcription-independent outgrowth of neurites in a clonal variant of PC12 cells (PC12D)
AU Sano, Mamoru
CS Aichi Human Service Center, Institute for Developmental Research, Aichi, 480-03, Japan
SO Current Topics in Neurochemistry (1997), 1, 27-40
CODEN: CTNEFZ
PB Research Trends
DT Journal; General Review
LA English
CC 2-0 (Mammalian Hormones)
AB A **review**, with 119 refs. Most studies of the NGF-dependent outgrowth of neurites have been performed with cultured sympathetic and sensory neurons or PC12 cells. The primary neurons have had prior exposure to NGF in vivo and they require NGF for basic survival. Although PC12 cells do not require NGF for survival, the outgrowth of neurites in such cells is a consequence of their differentiation into cells that resemble sympathetic neurons in response to NGF. Thus, the outgrowth of neurites is not necessarily a direct consequence of exposure to NGF. PC12D cells, a stable variant subcloned from native PC12 cell populations, produce neurites in a rapid transcription- and translation-independent manner upon exposure to NGF, bFGF, dbcAMP or staurosporine. The rapid sprouting of neurites occurs within minutes in local regions of PC12D cells that are exposed to NGF. Recent studies in conventional PC12 cells demonstrated the close relationship between the activation of MAP kinases (ERKs) and the outgrowth of neurites in response to various agents. Simultaneous activation and rapid **nuclear** translocation of MAP kinases were also observed in PC12D cells treated with NGF or bFGF. But the activation of MAP kinases was not observed in the outgrowth of neurites induced by dbcAMP or staurosporine. In this cell line, the NGF-dependent outgrowth of neurites was not blocked by the inhibition of the activation of MAP kinases by a MEK inhibitor, PD-98059. These results indicate that the activation of MAP kinases and subsequent expression of specific genes are required for the NGF-induced differentiation of PC12 cells but this pathway is not required for the NGF-dependent outgrowth of neurites. These processes have been investigated together in most studies of conventional PC12 cells. PC12D cells provide a unique exptl. system for studies of the cellular mechanism of the NGF-induced sprouting and elongation of neurites, sep. from the transcription-dependent differentiation of the cells.
ST **review** NGF transcription **neurite outgrowth**
PC12 neuron signaling differentiation
IT Animal cell line
(PC12; nerve growth factor-responsive and transcription-independent outgrowth of neurites in clonal variant of PC12 cells)
IT Nerve
(differentiation; nerve growth factor-responsive and transcription-independent outgrowth of neurites in clonal variant of PC12 cells)
IT Signal transduction, biological
Transcription, genetic
(nerve growth factor-responsive and transcription-independent outgrowth of neurites in clonal variant of PC12 cells)
IT Cell differentiation
(neuronal; nerve growth factor-responsive and transcription-independent outgrowth of neurites in clonal variant of PC12 cells)
IT Axon
(outgrowth; nerve growth factor-responsive and transcription-

independent outgrowth of neurites in clonal variant of PC12 cells)
IT 142243-02-5, Extracellular signal-regulated kinase
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(nerve growth factor-responsive and transcription-independent outgrowth of neurites in clonal variant of PC12 cells)
IT 9061-61-4, Nerve growth factor
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(nerve growth factor-responsive and transcription-independent outgrowth of neurites in clonal variant of PC12 cells)
RE.CNT 119 THERE ARE 119 CITED REFERENCES AVAILABLE FOR THIS RECORD
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independent outgrowth of neurites in clonal variant of PC12 cells)

IT 142243-02-5, Extracellular signal-regulated kinase
RL: BAC (Biological activity or effector, except adverse); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(nerve growth factor-responsive and transcription-independent outgrowth of neurites in clonal variant of PC12 cells)

IT 9061-61-4, Nerve growth factor
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(nerve growth factor-responsive and transcription-independent outgrowth of neurites in clonal variant of PC12 cells)

RE.CNT 119 THERE ARE 119 CITED REFERENCES AVAILABLE FOR THIS RECORD

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(4) Bar-Sagi, D; Cell 1985, V42, P841 CAPLUS
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ANSWER 6 OF 6 MEDLINE on STN
AN 96162646 MEDLINE
DN PubMed ID: 8581313
TI Multiple factors govern intraretinal axon guidance: a time-lapse study.
AU Brittis P A; Silver J
CS Department of Neurosciences, Case Western Reserve University, Cleveland, Ohio 44106, USA.
SO Molecular and cellular neurosciences, (1995 Oct) 6 (5) 413-32.
Journal code: 9100095. ISSN: 1044-7431.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199603
ED Entered STN: 19960327
Last Updated on STN: 19960327
Entered Medline: 19960319
AB In this study, the multiple factors that govern the unidirectional path of intraretinal axons, as well as the cellular movements prior to and during early axonogenesis, were investigated using time-lapse videomicroscopy. For several hours prior to overt axon elongation, young retinal ganglion cells send out transient minor processes in all directions at the pial surface. Time-lapse analysis of the chondroitin sulfate (CS)-containing matrix that has been suggested to play an important role in regulating this early differentiative event revealed the dynamic, wavelike properties of this extracellular matrix component. As the CS matrix dissipates across the immature ganglion cells, only one minor process, away from the highest concentration of CS peripherally and in the direction of the optic fissure centrally, is retained and becomes the mature axon. Focal concentrations of L1 appear at points of **neurite** contact with previously established axons, suggesting that this growth-promoting molecule is also involved with establishing the precise, unidirectional **outgrowth** pattern of retinal ganglion cell axons. NCAM was diffusely distributed on neural elements and on the neuroepithelial endfeet in the central and peripheral retina and, thus, may not be an essential unidirectional axon growth cue. Growth cones mechanically deflected 180 degrees from the optic fissure after the CS wave had receded from the central retina had morphologies and rates of elongation similar to those oriented in the proper direction. Growth cones deflected obliquely toward the ventral retinal periphery entered a territory of increasing CS-containing proteoglycan matrix and neurons with minor processes. As these deflected axons entered more deeply into this region they slowed down and sent out long transient branchlike processes. These observations illustrate the complex organization of the changing cell surface and matrix components within the retina during axonogenesis and axon **outgrowth**. The results also elucidate the potential importance of a cell state where immature neurons probe their environment via minor processes. These specialized **neurites** may provide the neuron with a way to sample a full 360 degrees of terrain around them. This method of exploring the environment could afford the cell a mechanism with which to sample, summate, and respond to physical structures as well as simultaneously occurring negative and positive molecular influences that are distributed unequally on either side of the **cell body**.
CT Check Tags: Female; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Animals
Axons: ME, metabolism
*Axons: PH, physiology
Axons: UL, ultrastructure
Image Processing, Computer-Assisted
Immunohistochemistry
Microscopy, Electron

ANSWER 6 OF 6   MEDLINE on STN
AN   96162646   MEDLINE
DN   PubMed ID: 8581313
TI   Multiple factors govern intraretinal axon guidance: a time-lapse study.
AU   Brittis P A; Silver J
CS   Department of Neurosciences, Case Western Reserve University, Cleveland, Ohio. 44106, USA.
SO   Molecular and cellular neurosciences, (1995 Oct) 6 (5) 413-32. Journal code: 9100095. ISSN: 1044-7431.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals
EM   199603
ED   Entered STN: 19960327
Last Updated on STN: 19960327
Entered Medline: 19960319

AB   In this study, the multiple factors that govern the unidirectional path of intraretinal axons, as well as the cellular movements prior to and during early axonogenesis, were investigated using time-lapse videomicroscopy. For several hours prior to overt axon elongation, young retinal ganglion cells send out transient minor processes in all directions at the pial surface. Time-lapse analysis of the chondroitin sulfate (CS)-containing matrix that has been suggested to play an important role in regulating this early differentiative event revealed the dynamic, wavelike properties of this extracellular matrix component. As the CS matrix dissipates across the immature ganglion cells, only one minor process, away from the highest concentration of CS peripherally and in the direction of the optic fissure centrally, is retained and becomes the mature axon. Focal concentrations of L1 appear at points of **neurite** contact with previously established axons, suggesting that this growth-promoting molecule is also involved with establishing the precise, unidirectional **outgrowth** pattern of retinal ganglion cell axons. NCAM was diffusely distributed on neural elements and on the neuroepithelial endfeet in the central and peripheral retina and, thus, may not be an essential unidirectional axon growth cue. Growth cones mechanically deflected 180 degrees from the optic fissure after the CS wave had receded from the central retina had morphologies and rates of elongation similar to those oriented in the proper direction. Growth cones deflected obliquely toward the ventral retinal periphery entered a territory of increasing CS-containing proteoglycan matrix and neurons with minor processes. As these deflected axons entered more deeply into this region they slowed down and sent out long transient branchlike processes. These observations illustrate the complex organization of the changing cell surface and matrix components within the retina during axonogenesis and axon **outgrowth**. The results also elucidate the potential importance of a cell state where immature neurons probe their environment via minor processes. These specialized **neurites** may provide the neuron with a way to sample a full 360 degrees of terrain around them. This method of exploring the environment could afford the cell a mechanism with which to sample, summate, and respond to physical structures as well as simultaneously occurring negative and positive molecular influences that are distributed unequally on either side of the **cell body**.

CT   Check Tags: Female; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Animals
Axons: ME, metabolism
*Axons: PH, physiology
Axons: UL, ultrastructure
Image Processing, Computer-Assisted
Immunohistochemistry
Microscopy, Electron

Rats
Rats, Sprague-Dawley
Retinal Ganglion Cells: ME, metabolism
*Retinal Ganglion Cells: PH, physiology
Time Factors

=>

Rats
Rats, Sprague-Dawley
Retinal Ganglion Cells: ME, metabolism
*Retinal Ganglion Cells: PH, physiology
Time Factors

=>

ANSWER 4 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN

AN 1985:520824 CAPLUS

DN 103:120824

ED Entered STN: 19 Oct 1985

TI The effect of exogenous gangliosides on neurons in culture: a morphometric analysis

AU Massarelli, R.; Ferret, B.; Gorio, A.; Durand, M.; Dreyfus, H.

CS Cent. Neurochim., CNRS, Strasbourg, 67084, Fr.

SO International Journal of Developmental Neuroscience (1985), 3(4), 341-8
CODEN: IJDND6; ISSN: 0736-5748

DT Journal

LA English

CC 13-6 (Mammalian Biochemistry)

AB Cultures of isolated neurons were treated with a purified preparation of gangliosides (10-5M and 10-9M) added to the cell growth medium at the 3rd day in culture, and a morphometric anal. of the cells was performed with an **image** analyzer after 1 and 4 days of treatment. The number of cells and the area of the **cell bodies** were increased following the treatment. Also, there was apparently a sprouting effect of the glycolipids on the number of secondary neuronal processes and an increase in the length of the primary **neurites**. The present data and other biochem. evidence (Dreyfus, H., et al., 1984) suggest that the addition of exogenous gangliosides may have a trophic effect on neurons, greatly enhances the number of cell-to-cell contacts, and possibly stimulates cell proliferation and differentiation.

ST neuron growth culture ganglioside; **neurite outgrowth** ganglioside

IT Nerve
(growth and morphol. of, in culture, gangliosides effect on)

IT Gangliosides
RL: BIOL (Biological study)
(neuron growth and morphol. and axon **outgrowth** in culture response to)

IT Animal tissue culture
(neuron growth and morphol. and axon sprouting in, gangliosides effect on)

IT Nerve
(axon, length of, in neurons in culture, gangliosides effect on)

ANSWER 4 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN

AN 1985:520824 CAPLUS

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ST neuron growth culture ganglioside; **neurite outgrowth** ganglioside

IT Nerve
(growth and morphol. of, in culture, gangliosides effect on)

IT Gangliosides
RL: BIOL (Biological study)
(neuron growth and morphol. and axon **outgrowth** in culture response to)

IT Animal tissue culture
(neuron growth and morphol. and axon sprouting in, gangliosides effect on)

IT Nerve
(axon, length of, in neurons in culture, gangliosides effect on)

ANSWER 3 OF 6 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1999:198128 CAPLUS
DN 131:16923
ED Entered STN: 29 Mar 1999
TI Effects of different fragments of tenascin-R on neuron morphology in vitro
AU Xu, Hanpeng; Xiao, Huasheng; Wang, Haojun; Liang, Zhe; Gong, Ju
CS The Institute of Neuroscience, Fourth Military Medical University, Xi'an, 710033, Peop. Rep. China
SO Journal of Medical Colleges of PLA (1998), 13(4), 272-275
CODEN: JMCPE6; ISSN: 1000-1948
PB Journal of Medical Colleges of PLA, Editorial Board
DT Journal
LA English
CC 13-6 (Mammalian Biochemistry)
AB To investigate the effects of different tenascin-R fragments on morphol. changes of neurons in vitro, cell suspension were prepared from E14-15 mouse embryo spinal cords by mech. dissection and trypsin digestion. The cells were cultured in dishes coated with different bacterial expressed tenascin-R fragments. After being cultured in serum-free medium for 26 h, the cells were fixed and stained by ABC immunocytochem. method for NSE. The cell number and **neurite** length were measured by a stereol. method using an **image** anal. system, and the data was analyzed statistically. The cells grew well in the serum-free medium for 26 h. Three types of cells were identified: (1) phase-bright cells with single or double **neurites**; (2) phase-dark cells with well branching **neurites**; (3) flat cells with 2-4 round vesicles in the **cell body** and radio-like **neurites**. The cell number and the **neurites** length were influenced by different tenascin-R fragments. It was found that FN1-2 fragment inhibited **neurite outgrowth**. Different tenascin-R fragments that were used as substrate exert varying effects on cultured neural cells, adhesion or anti-adhesion of cells, promotion or inhibition of the growth of **neurites**. These influences were mediated through receptors on the cell membrane. This study may provide some clues to the search for the search for the different receptors which may play essential roles in neuronal development and plasticity.
ST tenascin R neuron morphol
IT Nerve
(neuron; tenascin-R fragment effect on neuron)
IT Axon
(**outgrowth**; tenascin-R fragment effect on neuron)
IT Cell adhesion
Cell morphology
Spinal cord
(tenascin-R fragment effect on neuron)
IT Tenascins
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(tenascin-R; tenascin-R fragment effect on neuron)
RE.CNT 12 THERE ARE 12 CITED REFERENCES AVAILABLE FOR THIS RECORD
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     (FILE 'HOME' ENTERED AT 14:09:50 ON 16 MAR 2004)

     FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, CANCERLIT, JAPIO' ENTERED AT
     14:10:15 ON 16 MAR 2004
L1          19246 S (NEURITE OUTGROWTH)
L2             39 S L1 AND LUMINES?
L3             30 DUPLICATE REMOVE L2 (9 DUPLICATES REMOVED)
L4            731 S L1 AND REVIEW?
L5            477 DUPLICATE REMOVE L4 (254 DUPLICATES REMOVED)
L6             34 S L5 AND NUCLE?
L7             34 S L6 NOT L3
L8           3714 S NEURITE? AND (CELL BOD?)
L9             22 S L8 AND LUMINESC?
L10             7 S L9 AND IMAGE?
L11             4 DUPLICATE REMOVE L10 (3 DUPLICATES REMOVED)
L12            85 S L8 AND IMAGE?
L13            39 DUPLICATE REMOVE L12 (46 DUPLICATES REMOVED)
L14            39 S L13 NOT L11
L15             0 S L14 AND LUMINE?
L16             0 S L14 AND LUMINE?
L17             6 S L14 AND OUTGROWTH?

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     (FILE 'HOME' ENTERED AT 14:09:50 ON 16 MAR 2004)

     FILE 'BIOSIS, CAPLUS, EMBASE, MEDLINE, CANCERLIT, JAPIO' ENTERED AT
     14:10:15 ON 16 MAR 2004
L1         19246 S (NEURITE OUTGROWTH)
L2            39 S L1 AND LUMINES?
L3            30 DUPLICATE REMOVE L2 (9 DUPLICATES REMOVED)
L4           731 S L1 AND REVIEW?
L5           477 DUPLICATE REMOVE L4 (254 DUPLICATES REMOVED)
L6            34 S L5 AND NUCLE?
L7            34 S L6 NOT L3
L8          3714 S NEURITE? AND (CELL BOD?)
L9            22 S L8 AND LUMINESC?
L10            7 S L9 AND IMAGE?
L11            4 DUPLICATE REMOVE L10 (3 DUPLICATES REMOVED)
L12           85 S L8 AND IMAGE?
L13           39 DUPLICATE REMOVE L12 (46 DUPLICATES REMOVED)
L14           39 S L13 NOT L11
L15            0 S L14 AND LUMINE?
L16            0 S L14 AND LUMINE?
L17            6 S L14 AND OUTGROWTH?

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